

INTRODUCTION

Respiration

A. Concept

1. ***Respiration is not synonymous with cellular respiration***
2. In its complete meaning, consists of 4 distinct phases

B. Phases — essentially in order of occurrence

1. Breathing
 - a. Inspiration (inhalation) — beginning point
 - b. Expiration (exhalation) — end point
2. Gas Exchanges
 - a. Diffusion between inspired air & pulmonary capillaries
 - (1) O₂ diffuses from air in alveoli into pulmonary capillary blood
 - (2) CO₂ diffuses from pulmonary capillary blood into alveoli
 - b. Diffusion between systemic capillaries & tissue fluid
 - (1) O₂ diffuses from systemic capillary blood into tissue fluid
 - (2) CO₂ diffuses from tissue fluid into systemic capillary blood
 - c. Diffusion between all body cells & tissue fluid
 - (1) O₂ diffuses from tissue fluid into cells
 - (2) CO₂ diffuses from cells into tissue fluid
3. Gas transport
 - a. Blood transport of O₂ from pulmonary capillaries to systemic capillaries

- b. Blood transport of CO₂ from systemic capillaries to pulmonary capillaries
- 4. Cellular respiration [*no details -- from prerequisite*]
 - a. Concept — ATP production
 - b. Relation with other 3 phases
 - (1) Utilizes inspired, absorbed, transported O₂
 - (2) Generates waste CO₂ — transported, secreted, expired
 - c. 2 types
 - (1) Aerobic
 - O₂ utilized for oxidative phosphorylation
 - Most common
 - (2) Fermentation
 - Often termed “anaerobic” — incorrect
 - Oxidative phosphorylation with no O₂
 - Limited — liver, cardiac & some skeletal muscles

Respiratory System

A. Concept

Organs primarily or secondarily involved in breathing

B. Components & Functions

- 1. Nose & nasal passages
 - a. Warms incoming air — close to body temp.
 - b. Humidifies air — mucus binds water
 - c. Filters out particles

- (1) Hairs & sticky mucus
- (2) Cilia move mucus to throat — swallowed
- d. Olfaction
- e. Speech — sinuses contribute to voice resonance
- f. Anti-microbial
 - (1) Secreted bacteriostatic substances
 - (2) Mucus traps microbes
 - (3) Extensive sub-mucous layer with abundant lymphocytes & macrophages

2. Mouth

- a. Primarily a digestive organ, but can be an air intake
- b. Cannot effectively duplicate all of nose's functions

3. Pharynx (throat)

- a. Nasopharynx
 - Air passage
 - Lining same as nasal — similar functions
 - Openings from eustachian tubes of middle ears
 - Pharyngeal tonsils (adenoids)
- b. Oropharynx
 - Joins posterior mouth opening
 - Both air & food passage
 - Lining variable
 - Respiratory — pseudostratified
 - Digestive — non-keratinized stratified squamous
- c. Laryngopharynx

- Adjacent to laryngeal opening
- Inferior opening to esophagus
- Variable lining like oropharynx

4. Larynx (voice box)

a. Controls pharyngeal entry — glottis

- Open for air entry
- Closed during swallowing

b. Joins trachea

c. Phonation — vocal folds (cords) vary pitch

5. Trachea (windpipe)

a. Air passage only

b. Cartilage rings prevent collapse

6. Bronchial tree

a. General

- Progressively smaller branches (~25 levels) from trachea to eventually join the tremendous number of tiny air sacs
- Smooth muscle in walls to increase or decrease air flow [*details later*]

b. Bronchi

- Systematic branchings from trachea
- Logical divisions for right/left lungs, lung lobes & lobe subdivisions
- Irregular rings & patches of cartilage
- Pseudostratified lining

c. Bronchioles

- Lack cartilage — collapse prevented by air pressure

phenomena which keep air sacs open as well [*explained later*]

- Lining changes as diameter decreases, but always ciliated/goblet — eventually simple cuboidal

7. Alveoli (air sacs) [*details through rest of outline*]

- a. These are effectively the lungs
- b. They contain the air for gas exchange
- c. Tremendous collective capacity & surface area
 - 300 million
 - Total surface 75 M²
- d. Simple squamous walls for permeability

8. Skeletal muscles [*details later*]

- a. General — involved in mechanical forces to produce breathing movements
- b. Thoracic
 - External intercostals
 - Internal intercostals
 - Diaphragm
 - Minor role from some other muscles
- c. Abdominal — rectus abdominis

9. Thoracic skeleton

- a. General
 - Support for thoracic wall
 - Framework for muscular action
- b. Ribs
 - Primary importance

- Shape & attachments permit variable positions for breathing movements
- c. Sternum — anterior rib attachment point
- d. Vertebrae — posterior rib attachment points

MECHANICS OF BREATHING

Pressure Gradients

This is a basic requirement for breathing, since gases, like any other substance, will only move down a concentration gradient.

Inspiration

A. Introduction

1. Objective — induce outside air to enter respiratory passages & lungs
2. Gradient — one must be created so that lung pressure is less than atmospheric

B. Thoracic Expansion

1. Cause
 - a. General — size increase from muscle contraction & rib movements
 - b. Diaphragm
 - Contraction causes descent into abdominal cavity
 - Vertical increase in thoracic size
 - c. External intercostals
 - Contraction elevates ribs upward & outward

- Lateral & dorso-ventral increase in thorax

2. Effect

- a. Concept — intra-thoracic pressure decreases
- b. Location — space between visceral & parietal pleural membranes
- c. Reason
 - Thoracic cavity closed — no outside opening
 - Enlarged area from expansion
 - Gas pressure decreases when volume increases
- d. Amount
 - Quiet — from resting -5 mmHg to **-8 mmHg**
 - Forced — as much as **-40 mmHg**
- e. Pressure explanation
 - -5 mmHg (e.g.) means 5 below atmospheric
 - STPD (Standard Temperature and Pressure Dry) at sea level is 760 mmHg at 25°C — so, -5 mmHg is actually 755 mmHg

C. Lung Expansion

1. Cause

- a. General — size (alveolar capacity) increases
- b. Reason
 - Intra-thoracic pressure drop in surrounding space
 - Permits inflation from less pressure
 - Note that lungs are quite elastic, spongy & mostly filled with air

2. Effect

- a. Concept — intra-alveolar pressure decreases

- b. Reason — [same as intra-thoracic above]
- c. Amount
 - Quiet — from resting 0 mmHg to **-3 mmHg**
 - Forced — as low as **-35 mmHg**
 - [Note that intra-alveolar will always be 5 mmHg higher than intra-thoracic]**

D. Pressure Gradient

- Negative pressure in lungs is now below atmospheric of 0 mmHg within air passages & outside air surrounding head.
- Slight vacuum will suck air into expanded lungs
- Note — lungs & thorax do not expand due to air entry, but vice versa**

E. Surface Tension Effects

1. Concept — water creates cohesive tension at its surface with air
2. Positive effects
 - a. Location
 - Between parietal & visceral pleural membranes
 - Watery serous fluid secreted by membranes
 - b. Result
 - Moist membranes adhere to each other
 - Assists in lung expansion — resists pulling away from expanding thoracic wall
3. Negative effects
 - a. Location
 - Within alveoli
 - Moisture from secretions — creates necessary fluid

environment

b. Result

- This internally holds alveolar walls together
- Resists expansion

c. Solution

- Surfactant** secreted by special alveolar cells
- Acts as wetting agent — decreases surface tension

F. Lung Compliance

1. Concept — amount of stretch permitted
2. Cause — degree of elasticity
3. Importance
 - a. Determines effort needed for inspiratory expansion
 - b. Significant variable in forcefulness of inspiration
4. Pathological decrease
 - a. Cause — TB (e.g.) reduces elasticity by stiffening connective tissue around alveoli
 - b. Results
 - Lungs resist expansion
 - Extraordinary effort for lungs to follow expanding thorax

Expiration

A. Introduction

1. Objective — to force air out of the lungs & air passages
2. Gradient — opposite from that of inspiration, with intra-alveolar pressure higher than atmospheric

B. Quiet

1. Contrast with inspiration
 - a. At rest, all movements are **passive**
 - b. No muscular contraction required
2. Basic cause — inspiratory muscles merely relax
3. Results
 - a. Diaphragm
 - Intra-abdominal pressure was increased during inspiration from diaphragm pushing down
 - Pressure now forces up relaxed diaphragm
 - Not just to resting point prior to inspiration, but compression from muscle elasticity
 - b. Ribs
 - From relaxed external intercostals
 - Rib tension from expansion reversed
 - Thoracic compression
 - c. Lungs
 - Size decrease — elasticity permits
 - Surfactant
 - Aids in preventing complete collapse
 - Strong inner attraction of moisture
4. Intra-thoracic effect
 - a. Intra-thoracic pressure raised
 - b. Reason — air compression from thoracic compression
 - c. Amount
 - From peak inspiratory -8 mmHg up to **-2 mmHg**

- Increase of 6 mmHg
 - More than 3 mmHg inspiratory change
 - Due to acceleration from sharpness & force of diaphragm & thoracic wall recoil

5. Intra-alveolar effect

- a. Intra-alveolar pressure raised
- b. Reason – compression from intra-thoracic increase
- c. Amount
 - From peak inspiratory –3 mmHg to **+3 mmHg**
 - Same 6 mmHg increase as intra-thoracic

6. Pressure gradient

- Intra-alveolar pressure now above atmospheric of 0 mmHg in air passages & air surrounding head
- Alveolar air squeezed out of compressed lungs
- Note — thorax & lungs do not diminish due to collapse from air exit, but vice versa**

C. Forced

- 1. Active, contrasted with quiet expiration
- 2. Occurrences
 - a. Exercise
 - b. Hyperventilation
 - c. Sneezing
 - d. Coughing
 - e. Sighing

3. Causes
 - a. Basic
 - Muscle contraction
 - Different from inspiratory muscles
 - b. Internal intercostals
 - Different angle of attachment than external
 - Pull ribs downward & inward
 - Thoracic compression laterally & antero-posterior
 - c. Rectus abdominis
 - Pair of band-like muscles from ribs/sternum to pubis
 - Pulls down on ribs — enhances effect of internal intercostals
 - Greatly increases intra-abdominal pressure on diaphragm
4. Intra-thoracic effect
 - a. Increases thoracic pressure
 - b. Amount — to as much as **+40 mmHg**
5. Intra-alveolar effect
 - a. Increases intra-alveolar pressure — same extent as intra-thoracic
 - b. Amount — to as much as **+45 mmHg**
6. Pressure gradient
 - Much greater than during quiet expiration
 - More forceful expiration results

Volume Changes & Capacities

A. Introduction

1. Relates to lab work — spirometry
2. Subject to great individual variation
 - a. Inherent variables — e.g. sex & body size
 - b. Physical condition
 - c. Health

B. Tidal Volume (V_T)

1. Concept — amount inspired & then expired with each breathing cycle
2. Amount — normally 500 ml

C. Inspiratory Reserve Volume (IRV or complementary)

1. Concept — amount taken in by a maximum effort after inspiration of tidal
2. Amount — 2800-3000 ml

D. Expiratory Reserve Volume (ERV or supplemental)

1. Concept — amount expelled by a maximum effort after expiration of tidal
2. Amount — 1000-1200 ml

E. Residual Volume (RV)

1. Concept — amount remaining in lungs even after maximum forced expiration
2. Amount — 1200 ml
3. Cause — lungs partially stretched, never fully collapsed

F. Inspiratory Capacity (IC)

1. Concept — $IC = V_T + IRV$
2. Amount — $500 \text{ ml} + 3000 \text{ ml} = 3500 \text{ ml}$
3. Significance — maximum inspiration possible following expiration

G. Functional Residual Capacity (FRC)

1. Concept — $FRC = ERV + RV$
2. Amount — $1200 \text{ ml} + 1200 \text{ ml} = 2400 \text{ ml}$
3. Significance — maximum amount remaining at end of quiet expiration

H. Vital Capacity (VC)

1. Concept — $VC = V_T + IRV + ERV$
2. Amount — $4600/3800 \text{ ml}$ (men/women)
3. Significance — standard measure of respiratory fitness

I. Total Lung Capacity (TLC or Reserve)

1. Concept — $TLC = VC + RV$
2. Amount — $5800/5000 \text{ ml}$ (men/women)
3. Significance — maximum lung expansion possible

J. Ventilation (Minute Respiratory Volume or V_E)

1. Concept — quantity of air passing through lungs in one minute
2. Calculation
 - a. Formula — rate x volume
 - b. Resting — $14/\text{min} \times 500 \text{ ml} = 7 \text{ L/min}$
 - c. Maximum — $50/\text{min} \times 4500 \text{ ml} = 225 \text{ L/min}$
 - d. Minimum — $3/\text{min} \times 500 \text{ ml} = 1,500 \text{ ml/min}$

3. Variations — expected in different persons, due to sex, age & health
4. Significance — very practical application, since individual inspiratory/expiratory cycles & their volumes, though important, do not measure real life situation over time

K. Dead Space (V_D)

1. Concept
 - a. Volume of air in respiratory passageways
 - b. Within nostrils, pharynx, larynx, trachea, & bronchial tree
2. Amount
 - a. 150 ml
 - b. Constant for quiet or forced breathing
3. Significance
 - a. Does not function in gas exchange — only alveolar air can
 - b. First to be expired, so prevents complete expiration of air within alveoli
4. Physiologic dead space
 - Not the same concept as V_D
 - This is alveoli which are damaged & incapable of gas exchange

L. Alveolar Volume (V_A)

1. Concept — $V_A = V_T - V_D$
2. Amount — $500 \text{ ml} - 150 \text{ ml} = 350 \text{ ml}$
3. Significance — only this part of tidal volume is available for gas exchange

M. Alveolar Ventilation (V_A)

1. Concept — $V_A = \text{rate} \times V_A$
2. Amount — $14/\text{min} \times 350 \text{ ml} = 4.9 \text{ L/min}$
3. Significance
 - a. This reveals the effective minute respiratory volume, since it utilizes the alveolar volume
 - b. It is more accurate than V_E

Breathing Movements & Patterns

A. Introduction

1. These are standard situations
2. Their order is not significant
3. Most can be applied to the various test conditions utilized in lab

B. Eupnea

1. Ordinary quiet breathing
2. Occurs without awareness, with ease & comfort

C. Dyspnea

1. Labored distressed breathing
2. Exaggerated consciousness of necessity for increased effort
3. Occurrence
 - a. Normal — high level of exercise
 - b. Abnormal
 - Any blockage of airway or loss of exchange surface
 - e.g. — asthma or emphysema

D. Orthopnea

1. Dyspnea only when in a particular position
2. Always abnormal
3. e.g. — in congestive heart failure when lying down

E. Hyperpnea (Hyperventilation)

1. Increase in ventilation — usually rate & depth
2. Forced, but not distressed
3. Occurrence
 - a. Productive — exercise (below level that produces dyspnea)
 - b. Unproductive — pain or emotional stimuli

F. Bradypnea (Hypoventilation)

1. Decrease in ventilation
2. Always abnormal — e.g. shock

G. Polypnea (Tachypnea)

1. Increased rate with no depth increase
2. Always abnormal — e.g. thoracic/lung pain or during fever recovery

H. Apnea

1. Temporary absence of breathing
2. Occurrence
 - a. Normal — after non-productive hyperventilation, from lack of need due to excess O₂ & low CO₂
 - b. Abnormal — during heart attack [*see periodic below*]

I. Periodic

1. Not one particular type of breathing — recurring cycle of varied movements — i.e. pattern of different movements

2. **Cheyne - Stokes**

a. Most common

- Follows myocardial infarction
- At very high altitudes
- Some drugs — e.g morphine

b. Repeating pattern

- Very weak — bradypnea
- Hyperpnea
- Diminishing to apnea

GAS EXCHANGE

Introduction

A. Three Locations [*previously given*]

B. Gases [*previously given*]

C. Basis

Simple diffusion due to gas pressure gradients

Variables

A. Partial Pressure

1. Concept — in a gas mixture each exerts a pressure independently of the others, as though it alone occupied the total volume
2. This is an expression of the concentration of a gas

3. Symbolized by “P” before chemical symbol in smaller letters

B. Membrane Permeability

1. Involves alveolar, capillary & cell membranes
2. Exhibit variable permeabilities to respiratory gases
3. Will either enhance or inhibit exchanges

C. Chemical Reactions

1. Involves substances in blood, tissue fluid & cytoplasm
2. Concerned with either gas transport or cellular respiratory reactions
3. Efficiency of reactions exerts significant influence on efficiency of gas exchanges

D. Blood Circulation

1. Pulmonary & systemic pressure & flow influence efficiency of gas exchanges
2. Slow flow or low pressure would produce low partial pressures
3. Blood only available about 0.50 - 0.75 second each circuit

E. Alveolar Surface Area

1. Critical importance in permitting sufficient volume of gas exchanges
2. Inspired air only available for short time
3. Causes of reduction
 - a. Pneumonia
 - b. Emphysema

F. Air Volume

1. Ventilation of critical importance
2. Must vary proportionately during resting & active situations

3. Causes of reduction
 - a. Inappropriate rate &/or volume
 - b. Diseases — e.g. asthma or emphysema

Oxygen

A. Pulmonary

1. PO_2 of inspired air 160 mmHg (of total 760 mmHg)
2. Within alveoli PO_2 lowered to **100 mmHg** — due to continual diffusion out into blood
3. PO_2 of capillary blood **40 mmHg**
4. Diffusion of O_2 into blood perpetual — never reaches equilibrium
 - a. O_2 laden blood continually circulating out
 - b. Fresh 40 mmHg PO_2 blood continually being circulated in

B. Systemic

1. PO_2 of **100 mmHg** for freshly oxygenated blood circulating out of lungs back to heart
2. Remains at 100 mmHg through systemic capillaries
3. PO_2 of **40 mmHg** in tissue fluid surrounding capillaries
4. Diffusion of O_2 from capillaries into tissue fluid
5. PO_2 of tissue fluid raised to **100 mmHg**
6. Diffusion of O_2 into tissue fluid perpetual
 - a. O_2 continually diffusing into cytoplasm of cells, since continually expended in cellular respiration
 - b. Thus, effectively PO_2 of tissue fluid always 40 mmHg, despite continually gaining O_2 from blood
 - c. PO_2 of capillary blood effectively always 100 mmHg, due to fresh O_2 laden blood continually circulating in

7. PO_2 of **40 mmHg** in venous blood leaving capillaries
 - a. Remains until pulmonary capillaries, when cycle repeats
 - b. This is not severely O_2 depleted blood, since 75% saturated

C. During Exercise

1. Systemic
 - a. If strenuous, PO_2 can fall to **<5 mmHg** — only 15% saturated
 - b. Cause
 - Elevated cellular respiration
 - Leads to more diffusion from tissue fluid
 - Proportionately lowers PO_2 in systemic venous blood
2. Pulmonary
 - a. Greatly O_2 depleted incoming blood
 - b. Elevated pressure gradient
 - Enhanced diffusion from alveoli
 - PO_2 of 100 mmHg still achieved for outgoing blood

Carbon Dioxide

A. Systemic

1. Intracellular situation
 - a. CO_2 constantly generated by cellular respiration
 - b. PCO_2 of **46 mmHg**
2. Surrounding tissue fluid
 - a. PCO_2 of **40 mmHg**
 - b. Diffusion from higher intracellular PCO_2
 - c. Its PCO_2 elevated to **46 mmHg**

3. Systemic capillaries
 - a. PCO_2 of **40 mmHg**
 - b. Diffusion from higher tissue fluid PCO_2
 - c. Its PCO_2 elevated to **46 mmHg**
4. Perpetual concentration gradients
 - a. Venous capillary blood continually circulates out
 - b. Arterial capillary blood continually circulates in

B. Pulmonary

1. Entering blood still at PCO_2 of **46 mmHg**
2. Inspired alveolar air at PCO_2 of **40 mmHg**
3. Diffusion from blood into alveoli
 - a. Lowers blood PCO_2 to 40 mmHg
 - b. Elevates alveolar PCO_2 to 46 mmHg
4. Perpetual diffusion
 - a. Alveolar causes
 - CO_2 laden air continually expired
 - CO_2 depleted air continually inspired
 - b. Capillary causes
 - CO_2 depleted blood continually circulating out
 - CO_2 laden blood continually circulating in

GAS TRANSPORT

Oxygen

A. Hemoglobin (Hb)

[Structure previously given]

B. Reactions

1. O₂ specifically binds with iron (Fe) of heme unit
 - a. Forms oxyhemoglobin
 - b. Easily made bond — diffusion into pulmonary capillary blood is sufficient energy
2. O₂ release
 - a. Oxyhemoglobin very weak bond
 - b. Bond broken by energy gradient away from hemoglobin in systemic capillary blood
3. Roles of globin
 - a. Stabilizes Hb
 - b. Aids in reversibility of O₂ - heme reaction

C. Free O₂

1. Some O₂ dissolved in H₂O of blood
2. ~0.17ml [5ml with Hb]

Carbon Monoxide

1. Heme + CO = carboxyhemoglobin
2. Aggressive competitor
 - a. 230 times more affinity for heme than O₂
 - b. Much stronger bond than oxyhemoglobin
3. Very low P_{CO} in proportion to damaging effects
 - a. 0.4 mmHg will tie up 50% Hb as carboxyhemoglobin
 - b. May be lethal at P_{CO} of only 0.7 mmHg — this is only 0.1% of inspired air

Carbon Dioxide

A. Bicarbonate

1. 70% of CO₂ is transported in this form
2. Produced by dissociation of carbonic acid
 - a. CO₂ combines with blood's water to form carbonic acid, which ionizes to bicarbonate & hydrogen
 - b. $\text{CO}_2 + \text{H}_2\text{O} + \text{carbonic anhydrase} = \text{H}_2\text{CO}_3 = \text{HCO}_3^- + \text{H}^+$
3. Role of erythrocytes
 - a. Carbonic anhydrase
 - RBC enzyme — accelerates reaction between incoming CO₂ & cellular water
 - Reversible — CO₂ released to alveoli
 - Only tiny amount carried in plasma water — reaction too slow without this enzyme
 - b. Hemoglobin — buffers H⁺ to prevent pH upset

B. Carbaminohemoglobin

1. 20% of CO₂ is transported in this form
2. Combines with amino groups of globin
3. Small amount combines with plasma proteins
3. Slower reactions than bicarbonate

C. Free Solution

1. 10% of CO₂ is transported in this form
2. Dissolved in plasma in physical solution as free CO₂

REGULATION

Central Nervous Control

A. Introduction

1. Respiratory center
 - a. Within brainstem (lower brain)
 - b. 4 portions
 - 2 each in medulla & pons
 - Various interacting roles
2. Exact knowledge of mechanisms & interactions lacking

B. Medulla

1. Dorsal group
 - a. Interconnected group (pool) of neurons
 - Send impulses among each other continuously for certain time period
 - Impulses cease
 - Impulses restart
 - Rhythmic cycle continues
 - b. Nerves to inspiratory muscles
 - Cause contraction
 - Primarily for quiet inspiration
 - c. Quiet expiration from passive recoil during period of impulse cessation
 - d. Receives input from many peripheral receptors — *[details later]*
2. Ventral group
 - a. Utilizes same basic cyclic pool mechanism as dorsal

- b. Forced inspiration
 - Nerves from one portion to inspiratory muscles
 - Dorsal group active simultaneously
- c. Forced expiration
 - Nerves from separate portion to expiratory muscles
 - More pronounced action than inspiratory effect
- d. Apparently acts on signals from dorsal group

C. Pons

- 1. Pneumotaxic center
 - a. Essentially controls duration of inspiration
 - b. Continuous impulses to medulla
 - More frequent impulses cause more interruptions, so faster rate of breathing
 - Less frequent impulses cause fewer interruptions, so slower rate
- 2. Apneustic center
 - a. Much disputed in function & even its existence
 - b. Dorsal group effect
 - May control the depth of inspiration
 - Direct connection with dorsal group
 - Connections with pneumotaxic center, but can produce effect without this
 - c. Evidence from dysfunction
 - Connections to pneumotaxic & sensory input to dorsal group would have to be lost to isolate apneustic center
 - Severe inspiratory apnea results — prolonged inspiration with ineffective or no expiration

Sensory Influences

A. General Relations

1. All of these mechanisms interact with the neural regulators
2. They utilize receptors (sense organs) to monitor conditions which are affected by breathing rate & depth

B. Hering-Breuer Inflation Reflex

1. Stretch receptors
 - a. Within walls of bronchi & bronchioles
 - b. Detect degree of inspiratory expansion
2. Inspiratory effect
 - a. Over-inflation will cause increased activity
 - b. Inhibitory nervous impulses to dorsal group

C. Blood Pressure [*discussed with cardiovascular system*]

1. Baroreceptors
 - a. Within walls of aortic arch & carotid sinus
 - b. Monitor blood pressure
2. Vasomotor center
 - a. Within medulla — controls blood pressure
 - b. Signals from baroreceptors
3. Relation to breathing
 - a. Interconnections with respiratory center
 - b. BP influences efficiency of gas exchanges
 - c. Thoracic expansion & contraction directly affect BP

D. Chemoreceptors

1. Peripheral
 - a. Primarily within aortic & carotid bodies

- b. Not the same as baroreceptors
- c. Monitor blood PO_2 , PCO_2 & pH
 - More important for PO_2
 - Direct medullary response to PCO_2 & pH is more effective [*below*]
- d. Response
 - Low PO_2 stimulates receptors
 - Signals to respiratory center to increase breathing
- e. Amounts
 - No response until PO_2 falls below 60 mmHg — ventilation would double
 - Critical life-threatening level at 20-40 mmHg — ventilation increases 6x
- f. Relation to PCO_2
 - If PCO_2 increases PO_2 usually decreases
 - PCO_2 control responses will indirectly affect PO_2 , due to breathing increase
 - Thus, low PO_2 & peripheral receptor response would not be a strong influence if PCO_2 was increased as well

2. Medulla

- a. Receptors for both CO_2 & pH of blood
- b. Response
 - High PCO_2 & low pH stimulates
 - Breathing center stimulated in turn
- c. PCO_2 amounts
 - Arterial PCO_2 of only 50 mmHg will cause 4x increase in ventilation
 - PCO_2 of 80 mmHg will cause 10x increase

d. pH amounts

- Normal blood pH 7.4
- Maximum 4x ventilatory increase, at pH of 7.0
- Not as effective as PCO_2 , due to poor diffusion across blood-brain barrier

d. PCO_2 effects contrasted with PO_2

- More profound effect than changes in PO_2
- PCO_2 of 40 mmHg is only 0.5% saturation of blood, so changes are relatively of more consequence